CONSIDERATIONS ON THE PATHOLOGY AND THERAPEUTICS OP EPILEPSY.

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IF we scrutinize the accounts of those post-mortem examinations of epileptics which have been recorded in various portions of medical literature, we shall be astonished by the variety as well as by the inconstancy of the morbid anatomic changes. Almost every organ of the body has exhibited pathological modifications; but the variability of the majority of these phenomena precludes the possibility of designating any particular group of findings as especially characteristic of the disorder.

There is, however, one cerebral district, which in the majority of epileptics is the seat of more or less profound morbid changes—the cortex. In these cases the primary change may often be found in the cranial bones or in the membranes. In epileptiform attacks from gross morbid changes of the brain, the lesion is found situated in or near the vicinity of the cortex. Regarding the modus operandi of an epileptic attack, experimental investigation has furnished us with some suggestive data. Brown-Séquard has developed an epileptic condition in guinea-pigs by injury to different portions of the nervous system. The beautiful experiments of the renowned investigator in this department of research form one of the most classical chapters in experimental pathology.

Significant also are the experiments of Hitzig, by which the latter demonstrated that a state of artificial epilepsy may be induced by injury to certain portions of the cortex. Epileptic attacks have also been induced by passing an induction current through the cortex.

Again, Nothnagel has named a circumscribed spot on the floor of the fourth ventricle the "convulsion centre." He has found, that by irritation of this particular district the entire system of voluntary muscles may be thrown into a state of tonic and clonic spasm. While, however, irritation of this centre causes convulsions, it does not serve to explain the phenomenon of unconsciousness. Accordingly, an irritation of the adjacent vaso-motor centre is also assumed, which, by causing contraction of the arteries of the brain, results in cerebral anæmia, to which physiological circumstances Nothnagel attributes the loss of consciousness.

The anæmia of the first stage of the attack is followed by a very considerable venous hyperæmia; the latter condition serving to augment the irritability of the "convulsion centre," and to increase the duration of the convulsion. The venous hyperæmia is due to the convulsions of the cervical muscles, which exercise a constricting influence upon the large veins of the neck. This is certainly a most ingenious explanation.

Nevertheless, while cheerfully admitting the great prominence which the disturbances in the vaso-motor mechanism undoubtedly assume in the production of the attack, I cannot bring myself to believe, with Nothnagel, that all epileptic paroxysms are due to an irritation of what he terms the "convulsion centre." Rather would I accept that portion of Dr. Todd's theory, as elaborated by Hughlings

Jackson, which attributes the production of the convulsions to a discharge of accumulated nervous energy from the cortex. But admitting this to be indeed the case, our argument would not in the least exclude the possibility, in certain cases, of producing an epileptic attack by irritation of peripheral nerves or of the floor of the fourth ventricle.

I believe, however, that, as a rule, the clonic and tonic spasms are proximately due to a discharge from the cortex, consequent upon a primary decrepitude resident in, or capable (when elsewhere located) of indirectly deranging the functions of, the vaso-motor centre itself. What greatly confirms me in the belief that the vaso-motor system is more or less directly or indirectly invalidated in epilepsy, is not alone the extraordinary exhibition of circulatory anomalies, visible during the paroxysm, but also the derangements in peripheral circulation which these patients display during the interparoxysmal period. These circulatory disorders find an eloquent and unmistakable betrayal in the cold and cyanotic conditions of the extremities.

If asked to trace out chronologically to the best of one's ability those factors which are most intimately concerned in the production of an epileptic attack, I should do so somewhat as follows: (1) Derangement in the vaso-motor mechanism, culminating in anæmia; (2) unconsciousness due to excessive cerebral anæmia, and convulsions due to the irritative effects of the anæmia upon the cortex; (3) anæmia succeeded by venous engorgement, due to spasm in the cervical muscles and consequent prolongation of the convulsions owing to the irritative effects of the venous hyperæmia. That rapid cerebral anæmia will produce convulsions and unconsciousness has been inductively proven by Kussmaul and Tenner. These effects were obtained in animals by ligation of those arteries which supply the brain, as well as by general hemorrhage. Again, the cessation of the epileptic paroxysm, which takes place when the condition of venous hyperæmia, co-existing with the second stage of the attacks, is allayed by pressure upon the carotids, proves that the *continuance* of the convulsions, at least, was the co-sequence of the venous engorgement.

Respecting the scientific reasons for the similarity in the irritative effects of the two opposite conditions of excessive anæmia and excessive venous hyperæmia here referred to, I can offer no more adequate explanation than this: that these two antipodal conditions of the circulation are in so far identical in their physiological effects, as they both result in deficient aëration of the cerebral parenchyma. In sleep we have a *physiological* degree of anæmia, and in wakefulness a *physiological* degree of hyperæmia. It was doubtless a comparison of the vascular condition of the brain during the first portion of the epileptic attack with the condition of the same organ during sleep, which induced an eminent authority to speak of sleep as a "modified epilepsy." Such comparisons are, nevertheless, I believe, misleading, however trivially employed. The anæmia of sleep owes its origin entirely to physiological causes, while that of epilepsy is only attributable to a directly or indirectly induced morbidity in the functions of the vaso-motor centre.

Furthermore, anæmia is only *one* of other concomitant factors concerned in the causation of sleep, and to be physiologically effective must be accompanied by those same factors, more particularly by ganglionic exhaustion.

Respecting the reasons for the good after-effects which I have observed in patients whom I had treated for some time with prolonged instrumental compression of the carotids, I can only find an adequate explanation for this persistent improvement in an ameliorated condition of the vaso-motor mechanism consequent upon the rest and freedom from irritation afforded the same by the diminished arterial afflux.

I have for some time past treated these circulatory anomalies of epilepsy by compressing the carotids for protracted periods with an instrument which I designed for this purpose. *Vide* a paper read before the New York Neurological Society on "Carotid Compression and Physiological Brain Rest." Also an article published in the *Medical Record* of Feb. 18, 1882, as well as my monograph on the same subject published by Anson D. F. Randolph & Co., New York.

This improvement manifests itself in an increase of temperature in the extremities, in an evident improvement of the mental faculties, where these are impaired, and often in an absolute increase of weight. The beneficial effects of this treatment on innervation are certainly often most remarkable. It may perhaps be permitted, on this occasion, to venture a prediction as to the position which more or less protracted instrumental compression of the carotids may eventually assume in the treatment of this frightful disorder. I believe, then, that the most effective manner of employing it will be found to be in conjunction or alternating with very moderate doses of the bromides. Certainly a reduced and more philosophical employment of these powerful remedies is a consummation devoutly to be wished, and any procedure that holds out reasonable scientific hopes in this direction is eminently worthy of careful consideration.

This reduction is, however, only obtainable through the employment of some accessory agency, which shall aid and abet the bromides in the production of the physiological effects. Such an adjunct is, *par excellence*, more or less prolonged instrumental carotid compression. In employing this method of treatment, the physician should use his best diligence to apply compression *some time before the abnormal circulatory fluctuations transpire.* Where the prodromata of the attack are well marked, this is not dffiicult. Even should the aura be not well marked, and should the attacks be of irregular occurrence, treatment by carotid compression for from one to three and more hours each day will be found to render excellent therapeutic service. These good effects will soon be found to exhibit themselves in an improved condition of the peripheral circulation, as well as frequently in a marked improvement of the mental faculties, where these have become affected.

The more the constancy of a morbid habitude is invalidated, the less the tendency to a recurrence of the pathological manifestations progressively becomes, and consequently the prospects of eventual physiological restoration are proportionately increased.

The pervasiveness of this law is especially evident during the treatment of the periodical circulatory anomalies, which are so prominent and constant an accompaniment of certain convulsive affections, such as epilepsy, as well as of those conditions of morbid cerebral activity, as seen in insomnia, mania, etc.

Of one thing there can be no doubt: if we are ever to achieve permanent therapeutic benefits, we must not alone seek by soothing remedies to diminish the abnormal explosiveness of the cerebral plasma itself, but it should also be our endeavor to combat the morbid conditions prevalent in the domain of nutrition.

The most practical method of regulating the degree of plasmatic metamorphosis is by addressing our efforts to the blood-stream itself. To understand the absolute truth of this proposition, we have but to call to mind the extraordinary achievements of hydro-therapy, general faradization, and galvanism.